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Fluid responsiveness and changes in venous congestion and lung water during volume expansion in critically ill patients: a multicentre observational study

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Abstract

Background The relationship between fluid responsiveness and right-and left-sided congestion remains underexplored. This study aimed to delineate the dynamic behaviour of venous congestion and extravascular lung water index (EVLWI) during a fluid challenge depending on the concomitant changes in cardiac index (CI).

Methods In patients from three intensive care units, for whom a 500-mL fluid challenge was administered, we retrospectively analysed CI, central venous pressure (CVP), venous excess ultrasound (VExUS), and EVLWI, which had been prospectively recorded. A VExUS congestion point was calculated from 0 to 7 by assigning 1 point to each degree of abnormality for the 4 investigated veins. A subgroup analysis was planned in patients with acute respiratory distress syndrome (ARDS).

Results We analysed 64 patients, of whom 42 (66%) were fluid responders (FR+) defined by a CI increase ≥ 15% with fluid infusion. Before the fluid challenge, CVP was lower in FR + than in fluid non-responders (FR-) (7.3 [2.9–10.3] vs. 10.6 [8.2–13.0] mmHg,respectively, $p = 0.002$). VExUS grades and congestion points were not different between FR + and FR- (Grade 0: 62% vs. 55%, respectively, $p = 0.601$; congestion points: 1.0 [0.0–2.3] vs. 2.0 [1.0–3.0], respectively, $p = 0.053$). EVLWI was also similar between groups. Following fluid administration, VExUS grade deterioration occurred in 5% of FR + versus 73% of FR– ($p < 0.001$). After the fluid challenge, abnormal VExUS grades were more prevalent in FR – than in FR+ (91% vs. 43%, respectively, $p < 0.001$), and congestion points were higher (4.0 [3.0–5.0] vs. 1.5 [1.0–3.0],

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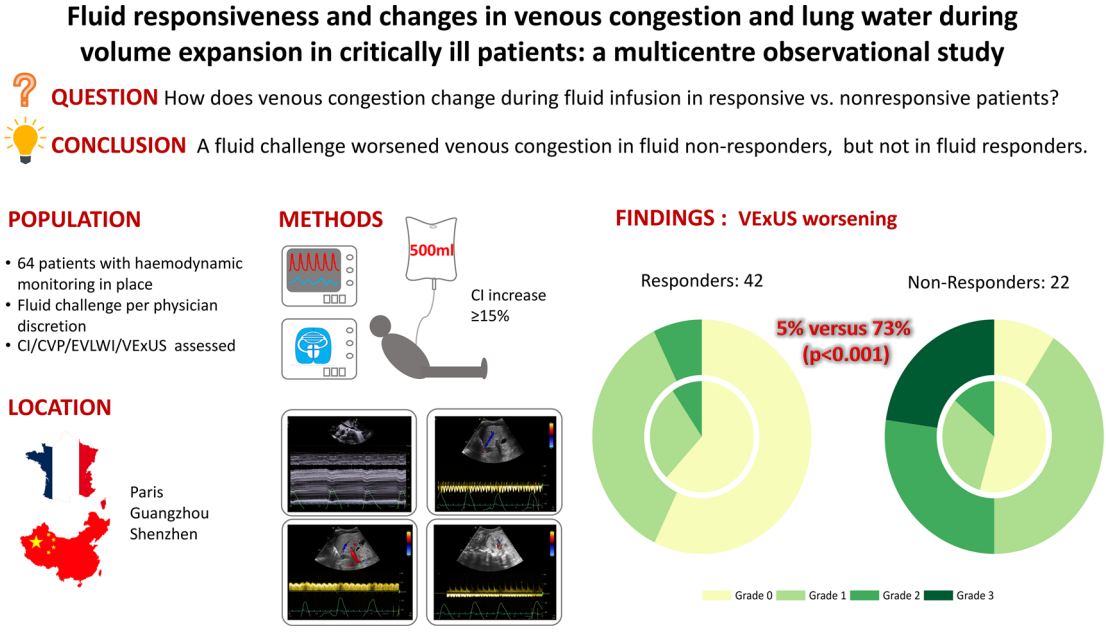
respectively, $p < 0.001$). CVP increased by 1.4 [0.4–2.4] mmHg in FR + and 2.0 [1.1–3.5] mmHg in FR- ($p = 0.064$). Among 25 patients with ARDS, EVLWI increased more than in patients without ARDS, in both FR- (by 1.7 [0.9–3.3] vs. 0.7 [0–1.4] mL/kg, respectively, $p = 0.046$) and FR+ (by 1.0 [-0.6–2.5] vs. 0 [-0.7–0.4] mL/kg, respectively, $p = 0.009$).

Conclusion A fluid challenge worsened venous congestion, assessed by VExUS as well as CVP, in fluid non-responders, while it was not in fluid responders.

Keywords Fluid resuscitation, Fluid challenge, Fluid tolerance, VExUS, Central venous pressure, Venous return, ARDS, Pulmonary vascular permeability, Echocardiography, Extravascular lung water.

Graphical abstract


Fluid responsiveness and changes in venous congestion and lung water during volume expansion in critically ill patients: a multicentre observational study
QUESTION How does venous congestion change during fluid infusion in responsive vs. nonresponsive patients?
CONCLUSION A fluid challenge worsened venous congestion in fluid non-responders, but not in fluid responders.
POPULATION
• 64 patients with haemodynamic monitoring in place
• Fluid challenge per physician discretion
• CI/CVP/EVLWI/VExUS assessed
METHODS
500ml
CI increase ≥15%
FINDINGS : VExUS worsening
Responders: 42
Non-Responders: 22
5% versus 73% (p<0.001)
Grade 0
Grade 1
Grade 2
Grade 3
LOCATION
Paris
Guangzhou
Shenzhen

Background

Volume expansion has long served as a cornerstone of treatment for patients with circulatory failure, particularly in the early phases of resuscitation (i.e., the salvage and optimization phases) [1]. Assessing fluid responsiveness, defined as the ability to significantly increase cardiac index (CI) in response to volume expansion [2], is recommended by the current guidelines to optimize fluid management [3–5]. However, fluid responsive status should not be interpreted as an obligatory trigger for fluid loading, as it should be weighed against the risk of excessive fluid administration [5, 6].

Fluid overload, which has garnered increasing attention [7], can be evaluated in two distinct compartments: the left side, which considers left heart filling pressures and the degree of pulmonary congestion, and the right side, which involves assessing right heart filling pressures and the extent of systemic venous congestion [8]. Markers of right-sided systemic congestion include central venous pressure (CVP) [9] and the Venous Excess Ultrasound (VExUS) score, which incorporates assessments of the inferior vena cava (IVC), hepatic, portal, and renal venous doppler patterns [10]. Left-sided pulmonary congestion can be evaluated using pulmonary artery occlusion pressure, or extravascular lung water indexed for ideal body weight (EVLWI) [11, 12] .

It was recently advocated to integrate the concepts of fluid responsiveness and venous congestion during the early resuscitation phase [13], given that both phenomena may coexist within the same patient [14]. Nevertheless, data delineating their potential relationship during volume expansion remain scarce so far. Notably, the two most recent studies were conducted in cardiac surgery patients, a cohort with a high prevalence of cardiac dysfunction, and primarily focused on the portal pulsatility index [15, 16]. Moreover, these investigations employed nonstandard fluid challenge protocols and lacked refined haemodynamic monitoring techniques to assess patient response.

The primary objective of this study was to describe the changes in right-sided congestion indices (VExUS and CVP) during fluid infusion according to fluid responsiveness. We hypothesized that, while fluid responsiveness and venous congestion may coexist, fluid responsive patients would be less likely to worsen venous congestion

than fluid unresponsive patients following volume expansion.

Patients and methods

We conducted a multicentre, observational study across three medical-surgical ICUs (Bicêtre hospitals, Assistance publique – Hôpitaux de Paris, France; First affiliated hospital of Sun Yat-sen University, Guangzhou, China; and Longgang central hospital, Shenzhen, China). It consisted of the retrospective analysis of data that were prospectively acquired in the context of daily care. The ethical review board of each participating institution approved this study (SRLF 22–033; FAH [2024] 436-1; LGH2025ECP-J027). Patients or their legally authorized representatives were informed about their participation and agreed to participate. The study was prospectively registered on ClinicalTrials.gov (NCT06772038).

Patients

Patients were eligible for inclusion if they met all of the following criteria: (i) age ≥ 18 y.o., (ii) infusion of a fluid challenge with 500 mL of crystalloid, as decided by attending physicians, (iii) central venous catheter in place with measurement of CVP, (iv) haemodynamic monitoring in place, utilizing either a transpulmonary thermodilution PICCO2 system or a pulse contour analysis ProAQT/PulsioFlex system (Pulsion Medical Systems, Getinge, Feldkirchen, Germany) and (v) measurement of VExUS performed at maximum 30 min before, and after fluid infusion.

Exclusion criteria included: (i) inadequate echographic window precluding a reliable VExUS assessment, (ii) presence of atrial fibrillation or significant arrhythmias, which prevent the acquisition of a stable venous doppler waveform, (iii) conditions interfering with portal vein flow assessment or interpretation (e.g., liver cirrhosis, portal hypertension, portal vein thrombosis), (iv) end-stage renal disease requiring maintenance dialysis, (v) pregnancy, (vi) extracorporeal membrane oxygenation, (vii) change in ventilatory parameters or catecholamine dosage during the study period and (viii) prone positioning.

Data collection

The study was conducted in accordance with the CODE-FIRE consensus [17]. Data retrospectively collected included demographic characteristics, admission shock category, use of vasoactive drugs, mechanical ventilation parameters, and severity scores. Clinically relevant outcomes such as ICU length of stay and 28-day mortality were also recorded. After the infusion of the fluid bolus, data were extracted from the bedside monitor through a USB stick, the PiCCO2 and Pulsioflex devices, and from the echocardiographs for post-hoc analysis.

Echocardiographic measurements

Echocardiographic measurements were obtained using either Vivid S6 (GE Healthcare, Madison, WI, USA) or Mindray M7 (Bio-Medical Electronics Co., Shenzhen, China). On the images that were retrospectively analysed, depending on the available views, we measured the left ventricular ejection fraction, tricuspid annular plane systolic excursion, and right ventricular dimensions, determined by the right-to-left ventricular end-diastolic area ratio.

Haemodynamic measurements

Cardiac index (CI) was measured by transpulmonary thermodilution [18, 19] and calibrated pulse contour analysis [20, 21], as detailed in the **Supplemental methods**. Variables derived from transpulmonary thermodilution included global end-diastolic volume index (GEDVI), extravascular lung water index (EVLWI), andpulmonary vascular permeability index (PVPI). CVP was measured at end-expiration (lowest values extracted from monitoring devices) [22]. Three CVP values were averaged for analysis.

Venous congestion assessment

All operators participating in the study were experienced intensivists certified in critical care ultrasonography. To ensure image acquisition consistency, all operators participated in an online meeting and joint training session before the study to standardize the VExUS acquisition (including measurement sites, acquisition procedures, probe type, Doppler settings, etc.). All VExUS measurements were reviewed at the Paris centre by an investigator (D.C.) who was blinded to the patient's condition and the fluid-responsive status. The measurements that were judged of poor quality, especially if no concomitant ECG tracing was present, were excluded. The VExUS grading system incorporates evaluations of the IVC and its independent components (hepatic, portal, and renal veins) [10], with measurements and grading described in **Supplemental methods**. On the acquired views extracted from the echograph, inter-observer reproducibility was evaluated through independent review of ultrasound images and videos by a blinded investigator (D.C.).

Given the overemphasized weighting of IVC in venous congestion grading [23, 24], we introduced VExUS organ points to refine assessment. Each vein was scored, with 0 assigned to an IVC < 2 cm and 1 assigned to ≥ 2 cm, while for the hepatic, portal, and renal veins, 0, 1, or 2 points were assigned to normal, mildly abnormal, and severely abnormal flow, respectively, yielding a total score of up to 7 points. Furthermore, venous flow was quantitatively assessed using hepatic S/D ratio, portal vein pulsatility index [25], and renal venous stasis index [26] (**Supplemental methods**). For each venous congestion variable,

three end-expiratory measurements were averaged for analysis.

Definition of fluid responsiveness and fluid tolerance

Fluid responsiveness was defined as an increase in CI ≥ 15% during the fluid challenge, in which patients received 500 mL of crystalloid over 15 min [18].

We defined fluid tolerance as a dynamic assessment of a patient's capacity to accommodate fluid administration without worsening organ congestion, rather than simply the absence of congestion per se (as congestion may pre-exist before volume expansion). Accordingly, fluid tolerance was defined as no deterioration in VExUS grade, no increase in VExUS points, and no increase in EVLWI following a fluid challenge.

Statistical analysis

The normality of data distribution was assessed visually. Normally distributed continuous variables were presented as mean ± standard deviation, whereas non-normally distributed data were reported as medians with interquartile ranges (IQRs, 25th-75th percentiles). Categorical variables were expressed as counts (n) and percentages (%). Inter-observer reproducibility was quantified a posteriori on echographic images using weighted Cohen's kappa statistic for categorical variables and intraclass correlation coefficient (ICC) for continuous variables.

The comparison of continuous variables between pre- and post-fluid challenge was performed with the Wilcoxon signed-rank test and visualized with paired boxplots, while categorical variables were compared using the McNemar test. Comparisons of variables in fluid responders and fluid non-responders were performed using either an unpaired Student's t-test or a Wilcoxon rank-sum test, depending on data distribution. Comparisons of proportions were assessed by either the chi-squared test or Fisher's exact test. An alluvial diagram was constructed to illustrate the transition of VExUS grade categories during fluid administration in fluid responders and fluid non-responders. Correlations between absolute changes (Δ) in haemodynamic and congestion variables during the fluid challenge were examined using the Spearman correlation coefficient and presented as a correlation matrix. As CVP and VExUS were measured before and after volume expansion in the same patients, their association was assessed using repeated-measures correlation (r_{rm}). A receiving operating characteristics (ROCs) curve was constructed to evaluate the ability of CVP to detect VExUS point. A subgroup analysis was planned in patients with acute respiratory distress syndrome (ARDS), according to the Berlin definition [27].

Sample size estimation was conducted to compare the evolution of VExUS grade between fluid responders and non-responders. We assumed that 10% of fluid responders would exhibit an increase in VExUS grade, compared to 50% of fluid non-responders. Given that tests predicting fluid responsiveness were used in some patients before infusing the fluid challenge, we expected a 2:1 ratio between fluid responders and non-responders. For an alpha of 0.05 and a power of 0.90, considering a possible dropout rate of 10%, we estimated a sample size of 64 patients.

All statistical analyses were performed using MedCalc software (version 20.218; MedCalc Software, Mariakerke, Belgium) and R (version 4.4.2; R Studio, version 1.0.136). A two-sided p-value < 0.05 was considered statistically significant.

Results

Patient characteristics

Of the 195 screened patients receiving volume expansion, 64 were ultimately included in the analysis (Figure S1). Each patient was included only once. Haemodynamic monitoring was conducted through transpulmonary thermodilution in 60 patients (94%) and with externally calibrated pulse contour analysis in 4 patients (6%). Following the 500-mL crystalloid infusion, 42 patients (66%) were fluid responders, while the other were fluid non-responders. The majority of patients had septic shock, and ARDS was present in 39% of the population (Table 1). The investigated volume expansions were conducted at 1 (1–3) days after ICU admission, with 40 patients (63%) evaluated within the first 24 h and 73% within 48 h. Echocardiographic images could not be retrospectively analysed, due to poor window or echogenicity, in 4 responders and 2 non-responders. Inter-observer agreement of the echo views, assessed by weighted kappa for VExUS grade and ICC for VExUS organ points, was 0.85 (95% CI, 0.71–0.99) and 0.91 (95% CI, 0.81–0.96), respectively.

Effects of volume expansion on congestion and other haemodynamic variables

Before the fluid challenge, CI, CVP, and GEDVI were significantly lower in fluid responders than in fluid non-responders (Table 2). Still at baseline, the VExUS grades and VExUS congestion points were similar between both groups (Table 3; Fig. 1). Overall, organ congestion was detected in 21 (55%) of the 38 patients with VExUS grade 0. Coexistence of fluid responsiveness and venous congestion occurred in 38% (16/42) of patients by VExUS grade and in 67% (28/42) by VExUS organ points (Figure S2). At the organ level, no significant differences were observed at baseline except for a larger portal vein pulsatility index in fluid non-responders than in fluid

Table 1 Patient characteristics in fluid responsive and non-responsive patients

Characteristic	Overall, *N* = 64	Fluid responders, *N* = 42	Fluid non-responders, *N* = 22	*p*-value[1]
Age (years), Mean ± SD	63 ± 13	65 ± 12	60 ± 14	0.127
Gender (male), n (%)	47 (73.4%)	32 (76.2%)	15 (68.2%)	0.491
SAPS II, Mean ± SD	64 ± 17	66 ± 16	60 ± 18	0.240
SOFA, Mean ± SD	8 ± 3	8 ± 2	9 ± 3	0.156
Lactate (mmol/L), Median (IQR)	2.2 (1.4, 3.1)	2.2 (1.5, 3.0)	2.1 (1.4, 4.4)	0.552
Serum albumin (g/L), Median (IQR)	28 (25, 31)	27 (24, 31)	30 (27, 31)	0.251
Cumulative fluid balance from admission to inclusion (mL), Median (IQR)	1766 (347, 2669)	1772 (205, 3893)	1755 (384, 2475)	0.572
Comorbidities, n (%)				
Chronic heart diseases	18 (28.1%)	12 (28.6%)	6 (27.3%)	0.913
Chronic heart failure	4 (6.3%)	3 (7.1%)	1 (4.5%)	> 0.999
Ischemic heart disease	12 (18.8%)	8 (19.0%)	4 (18.2%)	0.933
Valvular heart disease	2 (3.1%)	1 (2.4%)	1 (4.5%)	> 0.999
Hypertension	35 (54.7%)	24 (57.1%)	11 (50.0%)	0.586
Diabetes	37 (57.8%)	25 (59.5%)	12 (54.5%)	0.702
Chronic kidney disease	8 (12.5%)	6 (14.3%)	2 (9.1%)	0.704
Cancer	19 (29.7%)	12 (28.6%)	7 (31.8%)	0.787
Immunosuppressed	15 (23.4%)	8 (36.4%)	5 (22.7%)	0.728
Shock type, n (%)				0.392
Septic	48 (75.0%)	30 (71.4%)	18 (81.8%)	0.493
Pneumonia	20	12	8	
Abdominal infection	17	11	6	
Urinary tract infection	4	4	0	
Biliary tract infection	5	2	3	
Others	2	1	1	
Hypovolaemic	10 (15.6%)	8 (19.0%)	2 (9.1%)	
Cardiogenic	5 (7.8%)	4 (9.5%)	1 (4.5%)	
Obstructive	1 (1.6%)	0 (0%)	1 (4.5%)	
ARDS, n (%)	25 (39.1%)	13 (31.0%)	12 (54.5%)	0.078
Echocardiography				
LV ejection fraction (%), Mean ± SD	61 ± 11	61 ± 12	60 ± 11	0.758
RV/LV end-diastolic areas ratio, Mean ± SD	0.61 ± 0.16	0.61 ± 0.13	0.61 ± 0.20	0.921
TAPSE (mm), Mean ± SD	18 ± 5	18 ± 4	19 ± 5	0.313
Catecholamines				
Administration of norepinephrine, n (%)	57 (89.1%)	38 (90.5%)	19 (86.4%)	0.684
Norepinephrine tartrate dose (μg/kg/min), Median (IQR)	0.5 (0.2, 1.2)	0.7 (0.2, 1.2)	0.4 (0.2, 1.2)	0.358
Vasopressin or terlipressin infusion, n (%)	10 (15.6%)	6 (14.3%)	4 (18.2%)	0.726
Administration of dobutamine, n (%)	6 (9.4%)	5 (11.9%)	1 (4.5%)	0.655
Administration corticosteroids, n (%)	13 (20.3%)	6 (14.3%)	7 (31.8%)	0.114
Mechanical ventilation, n (%)	57 (89.1%)	36 (85.7%)	21 (95.5%)	0.406
Respiratory settings (in ventilated patients)				
Tidal volume (mL/kg PBW), Mean ± SD	6.1 ± 1.6	6.2 ± 1.5	6.0 ± 1.7	0.414
Plateau pressure (cm H_2O), Mean ± SD	19 ± 4	18 ± 4	19 ± 4	0.535
Positive end-expiratory pressure (cm H_2O), Mean ± SD	8 ± 3	8 ± 3	8 ± 3	0.673
PaO_2/FiO_2 (mmHg), Median (IQR)	275 (202, 345)	288 (209, 367)	250 (198, 286)	0.264
ICU length of stay (day), Median (IQR)	10 (7, 19)	9 (7, 19)	11 (7, 19)	0.476
28-day mortality, n (%)	17 (26.6%)	10 (23.8%)	7 (31.8%)	0.491

[1]Welch Two Sample t-test; Wilcoxon rank-sum test; Pearson's Chi-squared test; Fisher's exact test

SAPS II simplified acute physiology score II, SOFA sequential organ failure assessment, ARDS acute respiratory distress syndrome, LV left ventricle, RV right ventricle, TAPSE tricuspid annular plane systolic excursion, PBW predicted body weight, PaO_2/FiO_2 oxygen arterial partial pressure over inspired fraction of oxygen, ICU intensive care unit

Table 2 Evolution of haemodynamic variables before and after volume expansion in fluid responsive and non-responsive patients

	Before volume expansion	After volume expansion
Heart rate (beats/min), Median (IQR)		
Fluid responders (*n* = 42)	100 (87, 114)	99 (86, 107)
Fluid non-responders (*n* = 22)	108 (90, 117)	106 (86, 119)
Systolic arterial pressure (mmHg), Median (IQR)		
Fluid responders (*n* = 42)	119 (92, 140)	146 (128, 168) [ab]
Fluid non-responders (*n* = 22)	116 (101, 142)	115 (100, 147)
Diastolic arterial pressure (mmHg), Median (IQR)		
Fluid responders (*n* = 42)	52 (45, 57)	60 (56, 68) [a]
Fluid non-responders (*n* = 22)	55 (51, 76)	57 (52, 70)
Mean arterial pressure (mmHg), Median (IQR)		
Fluid responders (*n* = 42)	77 (68, 85)	93 (83, 98) [a]
Fluid non-responders (*n* = 22)	77 (67, 90)	79 (69, 97)
Central venous pressure (mmHg), Median (IQR)		
Fluid responders (*n* = 42)	7.3 (2.9, 10.3)	9.0 (4.7, 11.9) [a]
Fluid non-responders (*n* = 22)	10.6 (8.2, 13.0) [b]	13.1 (11.5, 15.0) [ab]
Cardiac index (L/min/m2), Median (IQR)		
Fluid responders (*n* = 42)	2.48 (1.86, 3.12)	3.21 (2.66, 4.09) [a]
Fluid non-responders (*n* = 22)	3.28 (2.75, 3.89) [b]	3.35 (2.76, 3.83)
GEDVI (mL/m2), Median (IQR)		
Fluid responders (*n* = 40)	576 (516, 711)	666 (589, 772) [a]
Fluid non-responders (*n* = 20)	729 (659, 858) [b]	780 (685, 959) [ab]
EVLWI (mL/kg PBW), Median (IQR)		
Fluid responders (*n* = 40)	8.0 (6.0, 10.2)	8.1 (6.0, 11.0)
Fluid non-responders (*n* = 20)	9.0 (7.4, 13.2)	10.0 (8.0, 14.5) [ab]
PVPI, Median (IQR)		
Fluid responders (*n* = 40)	2.1 (1.6, 2.7)	2.0 (1.4, 2.4)
Fluid non-responders (*n* = 20)	2.0 (1.6, 2.6)	2.2 (1.7, 2.9)

GEDVI global end-diastolic volume indexed for body surface, EVLWI extravascular lung water indexed, PBW predicted body weight, PVPI pulmonary vascular permeability index, IQR, interquartile range

[a] $p < 0.05$ after vs. before volume expansion, a: Wilcoxon signed-rank test

[b] $p < 0.05$ fluid responders vs. non-responders, b: Wilcoxon rank-sum test

Table 3 Evolution of venous congestion variables before and after volume expansion in fluid responsive and non-responsive patients

	Before volume expansion	After volume expansion
VExUS grade, n (%)		
Fluid responders(*n* = 42)		
Grade 0	26 (61.9%)	24 (57.1%)
Grade 1	12 (28.6%)	15 (35.7%)
Grade 2	4 (9.5%)	3 (7.1%)
Fluid non-responders(*n* = 22)		
Grade 0	12 (54.5%)	2 (9.1%) [ab]
Grade 1	7 (31.8%)	9 (40.9%)
Grade 2	3 (13.6%)	6 (27.3%)
Grade 3	0 (0%)	5 (22.7%)
VExUS organ points, Median (IQR)		
Fluid responders (*n* = 42)	1 (0, 2.3)	1.5 (1, 3) [a]
Fluid non-responders (*n* = 22)	2 (1, 3)	4 (3, 5) [ab]
IVC expiratory diameter (mm), Median (IQR)		
Fluid responders (*n* = 42)	16.2 (10.9, 18.2)	16.5 (13.4, 19.3) [a]
Fluid non-responders (*n* = 22)	18.0 (15.0, 21.6)	20.8 (18.0, 23.9) [ab]
Hepatic vein S/D, Median (IQR)		
Fluid responders (*n* = 42)	1.3 (0.9, 1.6)	1.4 (0.9, 1.7) [a]
Fluid non-responders (*n* = 22)	1.3 (0.8, 1.4)	0.9 (0.6, 1.3) [ab]
Portal vein pulsatility index (%), Median (IQR)		
Fluid responders (*n* = 42)	20.0 (13.1, 27.8)	21.9 (16.1, 29.6) [a]
Fluid non-responders (*n* = 22)	28.0 (24.4, 38.5) [b]	42.2 (33.7, 53.7) [ab]
Renal vein stasis index (%), Median (IQR)		
Fluid responders (*n* = 42)	9.8 (0.0, 19.2)	10.9 (0.0, 16.8)
Fluid non-responders (*n* = 22)	12.4 (3.6, 23.5)	27.3 (11.8, 42.3) [ab]

VExUS venous excess ultrasound, IVC inferior vena cava, IQR, interquartile range

[a] $p < 0.05$ after vs. before volume expansion, a: Wilcoxon signed-rank test, McNemar test

[b] $p < 0.05$ fluid responders vs. non-responders, b: Wilcoxon rank-sum test, Fisher's exact test

responders (Table 3). EVLWI was similar among groups (Table 2).

In the 42 fluid responders, volume expansion increased CI by 33 (21–40)%, and GEDVI by 15 (8–22)% (Table 2). There was no significant change in the distribution of VExUS grades (Table 3; Figs. 1 and S3). Among them, 95% showed no worsening of venous congestion, while about 30% had a 1-point VExUS increase. CVP increased significantly by 1.4 (0.4–2.4) mmHg, and the VExUS organ congestion points by 0 (0–1) (Table S1, Fig. 2). The evolution of venous congestion for individual organs is detailed in Table 3. In fluid responders, EVLWI remained unchanged compared to before the fluid challenge (Table 2).

In the 22 fluid non-responders, volume expansion increased CI by 4 (-7–8)%, and GEDVI by 3 (0–11)% (Table 2). The VExUS grade distribution changed significantly toward higher scores ($p = 0.003$). Grade 0 declined from 55% to 9% ($p = 0.002$) while grades 2 and 3 increased from 14% to 50% ($p = 0.006$) (Table 3; Fig. 1). VExUS grade deterioration was observed in 16 of 22 patients

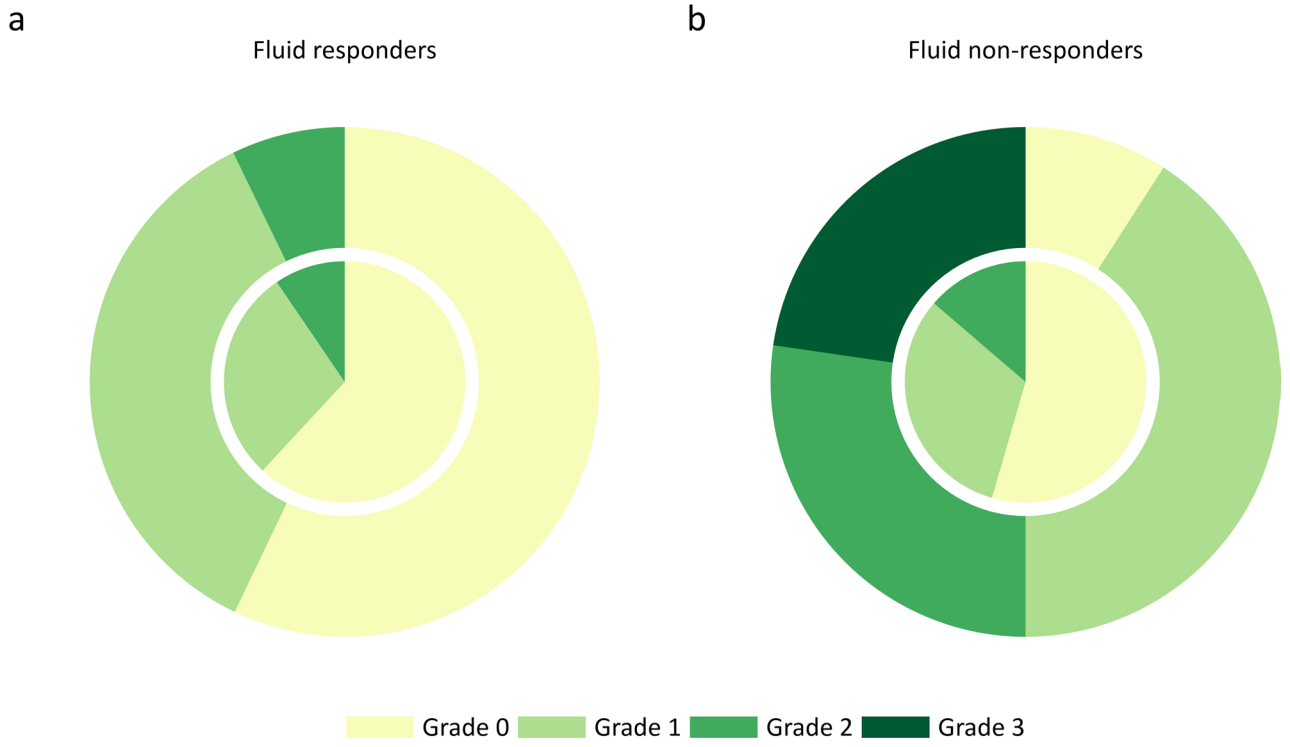


Fig. 1 Comparison of VExUS grading and its dynamic changes following a fluid challenge in FR+ (**a**) and FR- (**b**) patients. The inner circle represents pre-fluid challenge VExUS grade, and the outer circle represents post-fluid challenge VExUS grade. (**a**) FR+: pre- vs. post-, $p = 0.758$; (**b**) FR-: pre- vs. post-, $p = 0.003$. Inner circle: FR + vs. FR-, $p = 0.816$; Outer circle : FR + vs. FR-, $p < 0.001$. VExUS venous excess ultrasound, FR+: fluid responsive, FR−: fluid unresponsive

(73%) ($p < 0.001$ vs. fluid responders) (Figure S3). CVP increased significantly by 2.0 (1.1–3.5) mmHg ($p = 0.064$ vs. fluid responders) (Table S1, Fig. 2). The VExUS organ congestion points increased by 2 (1–2), and this change was larger than in fluid responders ($p < 0.001$) (Table S1, Fig. 2). The VExUS points significantly increased across all organs (Table 3). EVLWI significantly increased by 11 (4–20)%, to a larger extent than in fluid responders ($p = 0.011$) (Table 2).

After the fluid challenge, in fluid non-responders compared to responders, abnormal VExUS grades were significantly more prevalent (91% vs. 43%, respectively, $p < 0.001$) (Fig. 1), the VExUS congestion points were higher (Fig. 2), and congestion was more pronounced in all organs (Figure S4). EVLWI significantly increased only in fluid non-responders (Table 2).

Relationship between fluid responsiveness and fluid tolerance

Considering both fluid-responders and non-responders, the fluid-induced ΔCI was negatively correlated with ΔVExUS (r = -0.50 [-0.29 – -0.65], $p < 0.001$) (Figure S5). Right ventricular function and ventilation settings were not associated with the fluid-induced VExUS response (Table S2). CVP was significantly correlated with VExUS points (r_{rm} = 0.62 [0.44–0.75], $p < 0.001$) (Figure S6). The best thresholds of CVP to detect VExUS points ≥ 2, ≥3, and ≥4 were 8, 9, and 10 mmHg, with corresponding areas under the ROCs of 0.78 (0.69–0.86), 0.75 (0.66–0.83), and 0.83 (0.75–0.90), respectively (Table S3,Figure S7).

In fluid-responders, the slope of ΔCI/ΔGEDVI during volume expansion was steeper than in non-responders (0.77 [0.39–1.0] vs. 0.08 [-0.63–0.33], respectively, $p = 0.011$) (Table S4, Fig. 2). By contrast, the slopes of ΔCVP/ΔGEDVI (1.6 [0.5–2.7] vs. 4.2 [1.5–17.8], respectively, $p = 0.005$) and ΔVExUS/ΔGEDVI (0 [0–0.2] vs. 4.7 [1.7–9.0], respectively, $p < 0.001$) were flatter in fluid-responders than in non-responders (Table S4, Fig. 2).

Subgroup analysis in patients with ARDS

Patients with ARDS ($n = 25$), all monitored with a PiCCO2 device, were more severely ill than those without ARDS ($n = 39$) (Table S5). At baseline, there was no significant difference in venous congestion between patients with and without ARDS, as assessed by either VExUS or CVP (Table S6). EVLWI was significantly higher in patients with ARDS than in the other group (10.0 [9.3–13.0] vs. 7.0 [6.0–8.6] mL/kg, respectively, $p < 0.001$), as was PVPI (2.8 [2.1–3.2] vs. 1.7 [1.3–2.0], respectively, $p < 0.001$).

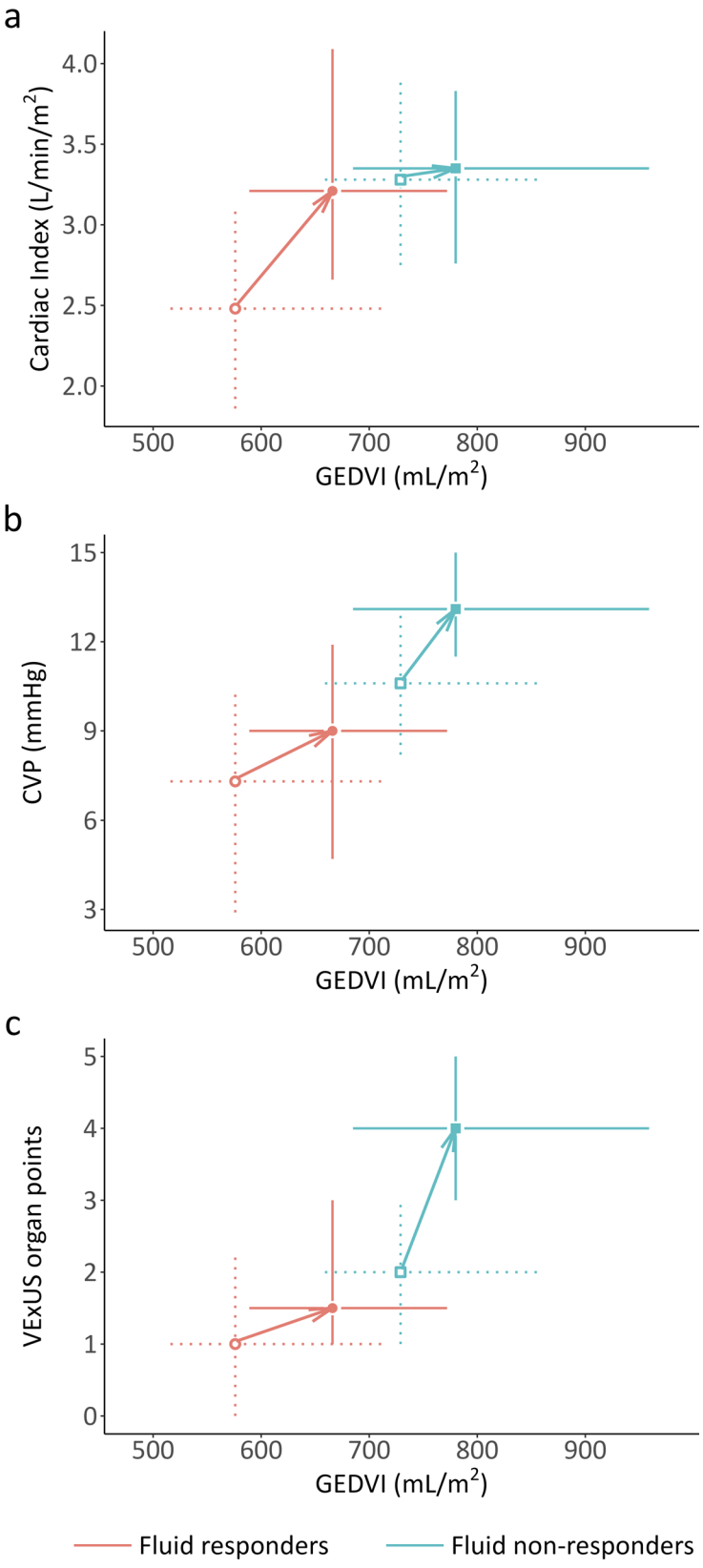


Fig. 2 (See legend on next page.)

(See figure on previous page.)

Fig. 2 The relationship between fluid responsiveness and venous congestion during volume expansion. (**a**) CI–preload (GEDVI) relationship in FR + and FR–, (**b**) CVP–preload (GEDVI) relationship in FR + and FR–, (**c**) VExUS–preload (GEDVI) relationship in FR + and FR–. The slopes represent the change in systemic flow or congestion per unit increase in preload. Dashed line: before fluid expansion, solid line: after fluid expansion. (**a**) CI slope: FR + vs. FR-, $p = 0.011$; (**b**) CVP slope: FR + vs. FR-, $p = 0.005$; (**c**) VExUS slope: FR + vs. FR-, $p < 0.001$. Data are expressed as medians with IQRs. CI: cardiac index, CVP: central venous pressure, VExUS: venous excess ultrasound, GEDVI: global end-diastolic volume index, IQRs: interquartile ranges, FR+: fluid responsive, FR–: fluid unresponsive

In the 13 fluid responders with ARDS, volume expansion increased CI by 34 (28–42)%, which was similar to the extent in responders without ARDS ($p = 0.324$) (Table S7). Changes in CVP and VExUS points were comparable between fluid responders with or without ARDS (ΔCVP: $p = 0.351$; ΔVExUS: $p = 0.747$). Among fluid responders, EVLWI increased significantly more in patients with ARDS (1.0 (-0.6–2.5) mL/kg) than in patients without ARDS (0 [-0.7–0.4] mL/kg, $p = 0.009$) (Table S8, Fig. 3).

In the 12 fluid non-responders with ARDS, CI increased by 5 (-5–8)%, with no significant difference compared to non-responders without ARDS ($p = 0.227$) (Table S7). Changes in CVP and VExUS points were similar between the two groups (ΔCVP: $p = 0.615$; ΔVExUS: $p = 0.500$). Among fluid non-responders, EVLWI increased significantly more in patients with ARDS (1.7 [0.9–3.3] mL/kg) than in patients without ARDS (0.7 [0–1.4] mL/kg, $p = 0.046$) (Table S8, Fig. 3).

Discussion

In the current study, we observed that (i) fluid responsiveness and venous congestion coexisted in some patients with circulatory failure; (ii) a fluid challenge worsened venous congestion to a greater extent in fluid non-responders than in fluid responders; (iii) CVP and VExUS were correlated, suggesting they both reflect venous congestion, and CVP, being simpler to measure, may be sufficient to assess right-sided congestion; and (iv) in patients with ARDS, EVLWI increased to a greater extent after a fluid bolus than in patients without ARDS.

In ventilated patients with circulatory dysfunction, Muñoz et al. reported that the prevalence of venous congestion, assessed by VExUS, was high and independent of the fluid responsiveness status. In this study, this status was assessed through a specific test but without giving fluid [14]. In our study, most patients were in the fluid optimization phase of circulatory failure within 1 (1–3) days of ICU admission, with relatively normal blood lactate, mean arterial pressure, and CI values, while almost all of them still required moderate to high doses of norepinephrine. Before fluid infusion, CVP was higher in fluid non-responders, as was the portal vein pulsatility index, and the VExUS congestion points tended to be higher. However, there was a large overlap of these variables in fluid responders and non-responders, confirming previous results with CVP [28].

After fluid infusion, we observed that the great majority of fluid-responsive patients (95%) exhibited no worsening of left- or right-sided congestion following fluid administration. In contrast, most fluid unresponsive patients (73%) developed worsening of right- and left-sided congestion. Regarding right-sided congestion, we found that approximately 30% of fluid responders presented with a 1-point increase in VExUS. These results confirm those suggested in more specific patients in our unselected critically ill patients [16]. The proportion of patients in whom VExUS increased during fluid infusion was lower than reported in a study in elective open-heart surgery patients, which employed the portal vein pulsatility index as the sole marker of venous congestion [16]. Another study, mainly involving post-cardiac surgery patients (78%), found that fluid challenges worsened venous congestion irrespective of fluid responsiveness [15]. Notably, the latter study exclusively enrolled patients with an IVC diameter > 2 cm, which may have introduced selection bias. Moreover, most patients enrolled in both latter studies had impaired cardiac function.

In fact, we believe that the larger fluid-induced worsening of right-sided congestion in fluid non-responders compared to responders is not surprising but reflects the physiological response to fluid infusion. In the case of fluid responsiveness, fluid infusion increases the mean systemic filling pressure (the upstream pressure of venous return) to a larger extent than it increases the right atrial pressure [29, 30]. The pressure gradient increases, so that venous return and cardiac output increase. In fluid non-responders, mean systemic filling pressure increases to the same extent as in fluid responders [29, 30]. However, cardiac output increases to a minimal extent, and congestion appears in the cardiac cavities. The right atrial pressure thus increases more than in responders and opposes the increase in mean systemic filling pressure. Hence, venous return and cardiac output change little or not at all. In other words, right atrial pressure, CVP, and venous congestion must increase to a larger extent in fluid non-responders than in fluid responders. "Fluid intolerance" in non-responders is inherent to the physiological effects of a fluid bolus, which have already been well described [30, 31].

On the contrary, on the left side, the lung congestion reflected by EVLWI is not inherent to fluid unresponsiveness, as it also depends on pulmonary permeability [11]. Once pulmonary permeability is impaired, as is typically

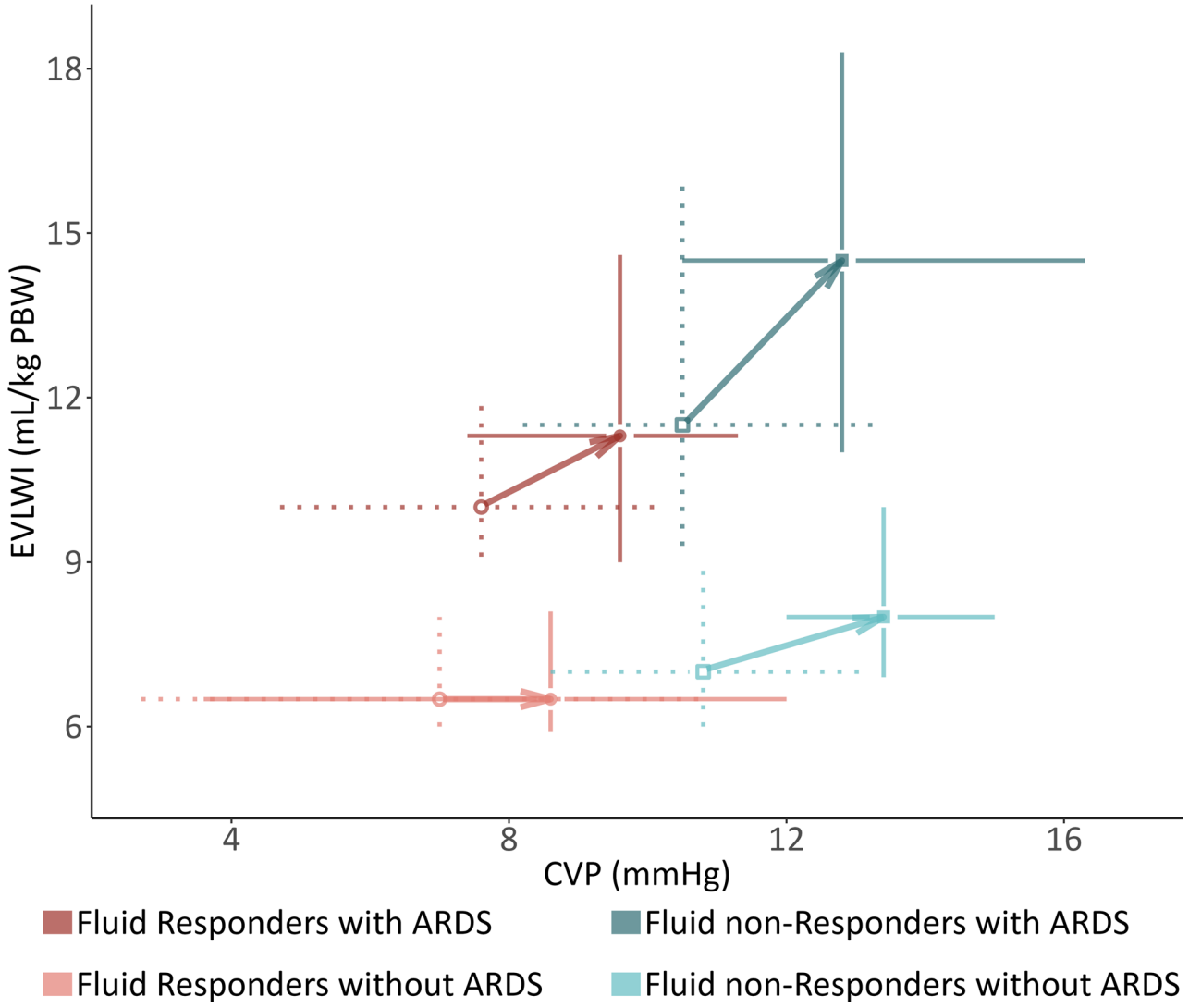


Fig. 3 The relationship between fluid responsiveness and lung water during volume expansion in ARDS versus non-ARDS patients. EVLWI increase depends on fluid responsiveness and pulmonary vascular permeability. Dashed line: before fluid expansion, solid line: after fluid expansion. Fluid responders: ARDS vs. non-ARDS, $p = 0.009$; Fluid non-responders: ARDS vs. non-ARDS, $p = 0.046$. Data are expressed as medians with IQRs. ARDS: acute respiratory distress syndrome, EVLWI: extravascular lung water indexed for ideal body weight, CVP: central venous pressure, IQRs: interquartile ranges

the case in ARDS, an increase in EVLWI can occur even when filling pressures are comparable to those in non-ARDS patients. In ARDS patients with elevated PVPI, lung water accumulation was more pronounced than in patients without ARDS. Furthermore, due to high PVPI and ongoing pulmonary capillary leak, EVLWI continued to rise significantly after fluid loading, even in fluid responders. These findings underscore the paramountcy of assessing fluid responsiveness before volume expansion in patients with ARDS [5].

We observed a similar evolution of CVP and of VExUS during the fluid challenge. In the original study by Beaubien-Souligny et al. [10], VExUS predicted the occurrence of renal failure following cardiac surgery better than CVP. However, this superiority was not retrieved in a subsequent meta-analysis in critically ill patients [32]. In such patients, our study suggests that measuring CVP could be sufficient to assess right-sided congestion and simpler to measure than the VExUS score.

A novel aspect of our study is the introduction of VExUS organ points, which offers two advantages compared to the conventional VExUS grading system. First, this approach mitigates the disproportionately high weight of IVC diameter in the grading system, acknowledging that an IVC diameter of < 2 cm does not preclude organ congestion [24]. In our cohort, among the 38 patients with an IVC < 2 cm, 55% (21/38) exhibited organ congestion. Conversely, only one patient with an IVC > 2 cm showed no organ congestion. The second advantage is that VExUS organ points are more

sensitive than conventional grading in detecting organ congestion. For instance, while the distribution of VExUS grades remained unchanged during fluid infusion in fluid responders, the VExUS organ pointsincreased significantly.

We acknowledge several limitations of our study. First, the dichotomization of fluid responsiveness may be criticized, as it represents a continuous physiological variable rather than a binary distribution. However, this classification addresses the research question through a pragmatic lens. Second, we did not follow CVP, VExUS, and EVLWI in the hours following fluid infusion. Then, we cannot state whether the changes we observed in fluid non-responders persisted in time. Third, all patients underwent advanced haemodynamic and ultrasound monitoring, with VExUS assessment requiring specialized skills (e.g., altered hepatic flow due to tricuspid regurgitation, intrarenal waveforms challenging to capture). This limits extrapolation to settings without such resources. Fourth, many patients were excluded from analysis as volume expansion performed in daily practice was not assessed with all the investigated variables. Finally, we did not explore the effects of venous and pulmonary congestion on clinical outcomes (e.g., AKI or mortality) [32], which warrants future investigations.

Conclusion

In critically ill patients with acute circulatory failure, a fluid challenge was associated with an impairment of venous congestion, assessed by VExUS and CVP, in fluid non-responsive patients, while it was not in fluid-responsive patients. This may reflect the normal behaviour of CVP in the case of preload unresponsiveness, while CVP, being simpler to measure, may be sufficient to assess right-sided congestion. These findings reaffirm that assessing fluid responsiveness should be a prerequisite for initiating fluid resuscitation and suggest that considering both fluid responsiveness and pulmonary permeability would be a way to personalize fluid therapy in patients at risk of fluid accumulation.

Abbreviations

CI	Cardiac index
CVP	Central venous pressure
EVLWI	Extravascular lung water index
FR+	Fluid responsive
FR–	Fluid unresponsive
GEDVI	Global end-diastolic volume index
IVC	Inferior vena cava
ICU	Intensive care unit
ICC	Intraclass correlation coefficient
IQRs	Interquartile ranges
PVPI	Pulmonary vascular permeability index
VExUS	Venous Excess Ultrasound

Supplementary Information

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Supplementary Material 1

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Author contributions

XS and XM had the idea of the study and conceptualized the research aims; XS and XM designed the study and took responsibility for the integrity of the data and the accuracy of the data analysis. XS, DC, RS, and WS implemented the study and collected the data; XS and DC did the statistical analysis and wrote the first version of the paper; RA, MB, AB, FB, FD, GLR, MF, ML, RLR, RFG, SL, RC, JW, and XG contributed substantially to the acquisition of data. TP, CL, and XM revised the first draft. All the authors approved the final manuscript. XM chaired the study.

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Data availability

The datasets used and/or analysed during the present study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The study protocol was approved by the Ethics Committee of each participating institutions (Bicêtre hospitals, No:22–033; First affiliated hospital of Sun Yat-sen University, No: [2024]436-1; Longgang central hospital, No:2025ECP-J027). The study was registered on ClinicalTrials.gov (NCT06772038). Informed consent was obtained from each patient or from the patient's legally authorized representative if the patient was unable to provide consent. The current study was performed in accordance with the Declaration of Helsinki.

Consent for publication

Not applicable.

Competing interests

XM is a member of the Medical Advisory Board of Pulsion Medical Systems (Getinge). He received honoraria for consultancy from Pulsion Medical Systems (Getinge), BD, Masimo and Baxter Healthcare. He received honoraria for giving lectures from AOP health, Pulsion Medical Systems (Getinge), BD, Philips, Masimo and Baxter Healthcare.

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